In the Specification:

Please amend the specification, without prejudice, as shown below. Deletions are indicated by crossing out, and additions are indicated by underlining.

Please replace the paragraph beginning on page 3, line 20, with the following amended paragraph:

Figures 3 A-E ~~depict GDNF-evoked calcium entry in Ret$^{-/-}$ DRG neurons.~~ **A.** In part of the DRG neurons, application of nominally Ca^{2+} free extracellular solution (no added EGTA) resulted in a delayed transient $[Ca^{2+}]_i$ elevation possibly due to activation of capacitative calcium entry (15 recorded neurons). This $[Ca^{2+}]_i$ overshoot was not observed when calcium concentration in the nominally Ca^{2+} free external solution was clamped to about 1 nM with 2 mM EGTA (14 recorded neurons; data not shown). A return to 2 mM external Ca^{2+} (wash-out) resulted in a pronounced $[Ca^{2+}]_i$ overshoot indicating an increased membrane permeability for Ca^{2+}. Switching back to nominally Ca^{2+} free external media in the presence of GDNF (100 ng/ml) resulted in a transient elevation of $[Ca^{2+}]_i$ with significantly prolonged kinetics of $[Ca^{2+}]_i$ decline. This indicates that at the resting membrane potential GDNF can prolong capacitative calcium entry either via more profound depletion of the internal stores or by a direct action on calcium channels in the plasma membrane. The traces are representative of 14 recordings performed in 3 independent experiments. **B.** GDNF was repeatedly applied both in the presence of the normal extracellular Ca^{2+} concentration and in the presence of nominally Ca^{2+} free external solution in Ret$^{-/-}$ DRG neurons. Application of GDNF (100 ng/ml) in the normal calcium extracellular solution evoked a typical long-lasting Ca^{2+} elevation which was reversed by wash-out. A switch to the nominally Ca^{2+} free external solution in the continuos presence of GDNF resulted in profound (and oscillatory in 5 out of 12 recordings) increase in $[Ca^{2+}]_i$ followed by slow decline in $[Ca^{2+}]_i$. Removal of GDNF (marked with arrow 1) led to a significant decline in $[Ca^{2+}]_i$. Readmission of GDNF led to quick elevation in $[Ca^{2+}]_i$ (arrows 2 and 3) (12 recorded neurons). Switching back to normal external Ca^{2+} containing solution resulted in an additional capacitative overshoot in $[Ca^{2+}]_i$. C. Pre-treatment with 10 μM U-73122 inhibited release of Ca^{2+} from the internal stores of Ret-negative DRG neurons and also in Ret-positive, but GFRα2-negative DRG neurons. **D.** The GDNF-evoked GPI linked protein-dependent sustained $[Ca^{2+}]_i$ elevation in Ret-negative

DRG neurons was abolished by pre-treatment with 1 U/ml PI-PLC. **E.** Shows effect of pre-treatment with 5 μM thapsigargin on GDNF-evoked $[Ca^{2+}]_i$ changes in wild type DRG neurons.

Please replace the paragraph beginning on page 11, line 13, with the following amended paragraph:

Figures 9 ~~A-B~~ A-C depict a schematic representation of the proposed Ret-independent GDNF-evoked signaling pathway. ~~**A.**~~ The GDNF-triggered membrane signaling most probably occurs within lipid rafts, as GFRα1 protein can be co-precipitated with Src type kinases in Triton X-100 insoluble membrane fractions. ~~**B.**~~ GDNF-evoked activation of GFRα1 induces Src type kinase (in particular, $pp62^{Yes}$ kinase in SHEP cells) activation and subsequent phosphorylation of PLCγ and MAP kinases. PLCγ activation leads to IP_3-dependent release of Ca^{2+} from internal calcium stores. Src-dependent phosphorylation of MAPK lead to its translocation to the nucleus and CREB activation.